Teaching alveolar ventilation with simple, inexpensive models

Stephen E. DiCarlo
Department of Physiology, Wayne State University School of Medicine, Detroit, Michigan

Submitted 23 June 2008; accepted in final form 7 August 2008

DiCarlo SE. Teaching alveolar ventilation with simple, inexpensive models. Adv Physiol Educ 32: 185–191, 2008; doi:10.1152/advan.90156.2008.—When teaching and learning about alveolar ventilation with our class of 300 first-year medical students, we use four simple, inexpensive “models.” The models, which encourage research-oriented learning and help our students to understand complex ideas, are distributed to the students before class. The students anticipate something new every day, and the models provide elements of surprise and physical examples and are designed to help students to understand 1) cohesive forces of the intrapleural space, 2) chest wall and lung dynamics, 3) alveolar volumes, and 4) regional differences in ventilation. Students are drawn into discussion by the power of learning that is associated with manipulating and thinking about objects. Specifically, the models encourage thinking about complex interactions, and the students appreciate manipulating objects and actually understanding how they work. Using models also allows us to show students how we think as well as what we know. Finally, students enjoy taking the models home to demonstrate to friends and family “how the body works” as well as use the models as future study aids.

pulmonary compliance; intrapleural cohesive forces; relaxation curves

HOW WE TEACH is much more important than what we teach because the traditional “lecture then test” format and accompanying “cookbook” laboratories discourage a deep understanding of the subject as well as the development of lifelong skills such as critical thinking, problem solving, communication, and interpersonal skills (5). This is important because in many schools, the classes are teacher centered rather than dynamic student-centered experiences that engage students in research-oriented learning. In this setting, the teacher perpetuates the process by transferring knowledge to students through lectures and note taking rather than active involvement and personal investment in the process (5). Teachers can encourage research-oriented learning by carefully considering the type and organization of information as well as the instructional strategies we use (5). For example, we all realize that reading the recipe is not as useful as preparing the meal when learning how to cook. How, then, do we change our approach from reading to “doing”? (5)?

Simple, inexpensive models encourage research-oriented learning and are often used to explain complex ideas because models promote logic, reasoning, and creativity (1, 2, 6–9, 15, 17–19). Physical models relate the unknown to the familiar and provide a new perspective on information gathering. Models also encourage a see-touch interaction to supplement new information processing.

Active participation with physical models can reach all types of learners via the visual, auditory, kinesthetic, and tactile schemes of learning (12, 21, 26), and manipulation of models is supported by constructivism (24). Constructivist learning theory is based on the idea that knowledge is actively constructed by the learner. Social constructivism suggests that knowledge creation is a shared experience rather than an individual experience (14). In this context, the important part is not that students manipulate things physically but that they do it for a purpose and engage in discussion about it (24). This is important because learning is a social process as well as an individual process (4).

When teaching and learning about alveolar ventilation with our class of 300 first-year medical students, we use four simple, inexpensive models. The models encourage research-oriented learning and help our students understand complex ideas. The models are distributed to the students before class and are designed to help students to understand 1) cohesive forces of the intrapleural space (Figs. 1 and 2), 2) chest wall and lung dynamics (Figs. 3 and 4), 3) alveolar volumes (Fig. 5), and 4) regional differences in ventilation (Fig. 6). Students are drawn into discussion by the power of learning that is associated with these experiences. The models encourage thinking about the complex interactions, and students appreciate manipulating objects and actually understanding how they work. Using models also allows us to show students how we think as well as what we know. Finally, students enjoy taking the models home to demonstrate to friends and family “how the body works” as well as use the models as future study aids.

We begin teaching about alveolar ventilation by asking “How does air get into the lungs?” Students are told that air flows from a region of higher pressure to a region of lower pressure by bulk flow. Accordingly, when alveolar pressure (P_A) is the same as the atmospheric pressure [barometric pressure (P_B)], no airflow occurs, because no pressure gradient exists.

Thus, to initiate air flow into gas exchange sites of the lung, P_A must decrease below P_B, or P_B must increase above P_A, as occurs during mechanical ventilation. To expel gases from alveoli, P_A must exceed P_B. With normal breathing, P_A must change because P_B does not fluctuate on a minute-to-minute basis.

How does P_A decrease below P_B? When the inspiratory muscles contract, the thoracic cavity and lungs enlarge, which decreases PA. Air then moves into the alveoli by bulk flow until PA and PB equalize at end inspiration.

Specifically, when the diaphragm contracts, its insertion is pulled toward its origin, flattening the dome, increasing the vertical dimension of the thoracic cavity (the thoracic cavity and lungs enlarge). Increasing the volume of the thoracic cavity reduces P_A below P_B. This concept is explained by Boyle’s law [Boyle’s law (P_1V_1 = P_2V_2)], which describes the inverse relationship between pressure (P) and volume (V; gas at a constant temperature). Increasing thoracic volume causes fewer collisions between gas molecules and between gas mol-
ecules and the thorax. The lower number of collisions decreases the pressure exerted by the gas, which reduces $P_A$ below $P_B$ so air flows into the lungs until $P_A$ again equals $P_B$ at end inspiration.

Once air enters the lungs, how does air get out of the lungs? The lung is stretched during inspiration, so when the inspiratory muscles relax, the lung recoils to compress the alveolar gas volume. This elevates $P_A$ above $P_B$ so air is expelled (expired) until $P_A$ again equals $P_B$ at end expiration. Specifically, when the diaphragm relaxes, its elasticity causes it to return to its dome shape, decreasing the vertical dimension of the thoracic cavity. Again, the lung is stretched during inspiration, so when the inspiratory muscles relax, the lung recoils to compress the alveolar gas volume. This elevates $P_A$ above $P_B$ so air is expelled (expired) until $P_A$ again equals $P_B$ at end expiration.

Importantly, this explanation, although correct, requires further elaboration (and this is when we introduce the model). Specifically, how does increasing the vertical dimension of the thoracic cavity expand the lungs?

To answer this question, students are told that the respiratory system can be modeled as an air-filled balloon surrounded by a larger water-filled balloon (10). The outside of the outer

water-filled balloon represent the atmosphere. The small liquid-filled compartment between the two balloons represents the pleural space and intra-pleural pressure. The air-filled compartment inside the inner air-filled balloon represents the alveoli and conducting airways of the lungs and intra-alveolar pressure.

Fig. 1. The respiratory system can be modeled as an air-filled balloon surrounded by a larger fluid-filled balloon. The outside of the outer fluid filled balloon represent the atmosphere. The small liquid-filled compartment between the two balloons represents the pleural space and intra-pleural pressure. The air-filled compartment inside the inner air-filled balloon represents the alveoli and conducting airways of the lungs and intra-alveolar pressure.

Fig. 2. The model used to facilitate the understanding of the cohesive forces of the intra-pleural space consists of two microscope slides with a few droplets of water (joined oval structures) placed between them. The slides move easily over one another horizontally; however, it is very difficult to pull them apart.

Fig. 3. The bony, moderately flexible ribcage can be modeled by cutting the corners of a wire coat hanger (top, the dashed lines indicate the location for cutting the hanger into two chest cage models; note that two models are obtained from each hanger). The model used to simulate the lung is a rubber band (bottom, shaded circle). The lungs inside the chest cavity, with the respective pleural surfaces held together by cohesive forces, are simulated by placing the rubber band around the arms of the coat hanger (1). The volume of the lung is higher than its equilibrium volume as simulated when the rubber band is stretched open (2). Chest cavity volume is less than its equilibrium volume as simulated when the ends of the hanger are compressed (3). The equilibrium volume of the combined lung-chest cage represents a point [functional residual capacity (FRC)] where the tendency of the lung to deflate (rubber band to collapse, inward pointing arrows) is balanced by the tendency of the chest cage to expand (hanger to spring open, outward pointing arrow) (4). These equal but opposite recoil forces create an intrapleural pressure that is below atmospheric pressure. Furthermore, as a person inhales to expand the chest cage and lung, intrapleural pressure becomes increasingly subatmospheric as a consequence of the force tending to separate the lung and chest wall.
pleural, and the inside of the thorax is lined with a membrane called the parietal pleura. These pleural membranes juxtapose to form a pleural sac around the lung. The space within the pleural sac contains a few milliliters of fluid. The intrapleural fluid creates cohesive forces that hold the visceral and parietal pleura together. The cohesive forces opposes the tendency of the ribcage to expand (increase in size) and the tendency of the lungs to collapse (decrease in size) (10, 20). Opposing these directionally opposite forces creates the negative intrapleural pressure. Intrapleural fluid also creates a slippery surface allowing the lungs to slide within the chest against the thoracic wall and, when the chest expands during inspiration, the lungs are compelled to follow so that the lungs and chest expand as a single unit.

This concept and physiological significance of the cohesive forces of the intrapleural space are often difficult for students to grasp. To help students understand this concept, we and many others use a simple model (19). The model used to facilitate the understanding of this concept consists of two

---

Fig. 4. Top: diagram of the lung and chest cage. Arrows show the movement of the chest cage and lung. Bottom: Separate relaxation curves for the lung (right) and chest cage (left) along with the combined lung-chest cage relaxation curve (middle). The combined lung-chest cage curve is the algebraic sum of the separate lung and chest cage curves. The slope of each relaxation curve corresponds to the compliance for the structure(s). At end expiration (point A), recoil or relaxation pressure for the lung and chest cage alone are equal but opposite. At this point, lung volume corresponds to FRC. As additional air volume is inhaled into the lung, the lung is stretched further and exhibits a greater recoil pressure. At the same time, the chest cage is less compressed, so its negative recoil pressure diminishes as it approaches its equilibrium volume. When a slightly larger air volume is inhaled, the chest cage reaches its equilibrium volume (0-mmHg relaxation pressure, point B), and the lung and lung-chest relaxation curves intersect (point C). Thereby, at this lung volume, all measured relaxation for the lung-chest cage system is from the lung because the chest cage was at its equilibrium volume (point B), or the volume it would assume if the lung were not present. If an even greater air volume is inhaled (point D), both the lung and chest cage are stretched beyond their equilibrium volumes. Note that the compliance curve for the combined lung-chest cage becomes more flattened (less compliant) at this point because the lung and chest cage are both tending to recoil toward smaller equilibrium volumes. If the total lung-chest cage system is returned to resting end expiration (point A) and air is expelled, a negative relaxation pressure results for both the chest cage and combined lung-chest cage (point E). At this point, the chest cage is compressed as more and more air is expelled, with the negative recoil pressure resulting from the tendency of the chest to expand toward its equilibrium volume (point B). At the same time, the lung contributes little positive relaxation pressure because it is close to its equilibrium volume (i.e., 0-ml volume) because it is stretched very little. Students are encouraged to manipulate their models as they discuss these relationships with the instructor. [From the Integrated Medical Curriculum (6a)].

Fig. 5. Alveolar ventilation is the volume of fresh air introduced into the gas-exchanging regions of the lungs per minute. Top: the model consists of a paper cutout of the anatomic dead space with the alveoli as well as two columns of paper, each with four segments, with each segment representing 150 ml of air. One column has three dark blue segments and one light blue segment, and the second column has three light blue segments and one dark blue segment. Dark blue segments represent atmospheric (fresh air), and light blue segments represent alveolar air (air that has previously equilibrated with pulmonary artery blood). The anatomic dead space and columns of air are sized such that one of the 150-ml segments fits perfectly into the anatomic dead space while the remaining three segments fit perfectly into the alveoli (bottom).
microscope slides with a few droplets of water placed between them (Fig. 2). The slides move easily over one another horizontally; however, it is very difficult to pull them apart. Similarly, intrapleural fluid creates a slippery surface allowing the lungs to slide within the chest against the thoracic wall. The intrapleural fluid also creates cohesive forces that hold the visceral and parietal pleura together. Thus, when the chest expands during inspiration, the lungs are compelled to follow so that the lungs and chest expand as a single unit.

Lung and Chest Wall Dynamics Model

The lung and chest wall dynamics model is based on that described in Refs. 22 and 23. Once this concept (i.e., lung and chest wall are held together by cohesive forces) is fully understood and demonstrated with the model, it becomes necessary to discuss the complex interactions between the lung and chest wall.

To do this, students are shown that the bony, moderately flexible chest cage is analogous to a punctured tennis ball (at this point tennis balls, previously cut in half, are tossed into the audience). Because the flexible ribcage is compressible but does not collapse, the chest cavity has considerable air volume when inside and outside pressure are equal, or transmural pressure is 0 mmHg. The model used to simulate the lung is a rubber band (Fig. 3, bottom). When no forces are acting on the rubber band, its equilibrium volume (represented by the area of the rubber band) is close to 0. However, the lung, like the rubber band, can be stretched, and the volume (area of the circle) increased.

When the rubber band is placed around the two arms of the corners of the coat hanger, the triangular area between the two arms of the corner of the coat hanger and the rubber band can be used to represent the volume of air in the lungs at a given point in time. Furthermore, the inwardly directed recoil force of the rubber band opposes the outwardly directed recoil force of the coat hanger (Fig. 3, bottom). When there are no forces acting on this model, the inwardly and outwardly directed recoil forces reach a balance, such that the inwardly directed force of the rubber band is equal and opposite to the outwardly directed force of the coat hanger. This point is analogous to the end of a quiet exhalation. The volume of air left in the lungs at the balance point is equal to the functional residual capacity (FRC).

At this point, contraction of the muscles of inspiration causes the chest wall to expand and the volume of the lungs increases. Similarly, pulling out on the arms of the hanger stretches the rubber band (increasing the volume) as well as increasing the inwardly directed recoil force of the rubber band while simultaneously decreasing the outwardly directed recoil force of the hanger (Fig. 3). Relaxation of the muscles of inspiration allows the increased recoil force of the lungs to drive quiet (passive) exhalation. Similarly, releasing the force on the arms of the hanger allows the model to return to its equilibrium position.

Thus, with the lungs placed inside the chest cavity and their respective pleural surfaces held together by cohesive forces (the rubber band placed around the arms of the coat hanger; Figs. 3 and 4), the volume of the lung is higher than its equilibrium volume (the rubber band is stretched open), whereas chest cavity volume is less than its equilibrium volume (the ends of the hanger are compressed). Again, the equilibrium volume of the combined lung-chest cage represents a point at which the tendency of the lung to deflate (rubber band to collapse) is balanced by the tendency of the chest cage to expand (hanger to spring open). Specifically, at the equilibrium volume of the combined lung-chest cage, the lung (rubber band) is expanded above and the chest cage (hanger) is compressed below their respective equilibrium volumes (Fig. 3, bottom). The equilibrium volume of the combined lung-chest cage corresponds to resting end expiration, or the position (volume) the combined lung-chest cage would assume when the respiratory muscles are relaxed. At resting end expiration, the recoil pressure of the lung tending to deflate (rubber band collapse) is opposed to an equal but opposite recoil pressure of the chest wall tending to expand (hanger springing out; Fig. 3). These equal but opposite recoil forces create an intrapleural pressure that is below atmospheric pressure. Specifically, a “tug of war” between the lung and chest wall increases the intrapleural space and decreases
the intrapleural pressure (13). Furthermore, as a person inhales to expand the chest cage and lung, intrapleural pressure becomes increasingly subatmospheric as a result of the force tending to separate the lung and chest wall.

The hanger and rubber band model can be used to understand changes that occur with obstructive and restrictive pulmonary diseases. For example, many restrictive pulmonary diseases result in a decreased compliance of the lung and/or chest. To model this condition, simply add a second or thicker rubber band to the hanger (22, 23). This situation reduces equilibrium volume, makes inspiration more difficult, and requires more muscular effort to move the chest wall (spread the arms of the hanger) and inflate the lungs (stretch the rubber bands).

Some obstructive pulmonary diseases result in an increased compliance of the lungs. This can be demonstrated by using a thinner rubber band than was used for the normal state (22, 23). With the thinner rubber band, inspiration is easier because the lungs are more compliant. In addition, the inwardly directed recoil force of the lungs, used to drive exhalation, is reduced, resulting in a prolonged expiration. Finally, a “new” equilibrium point is found between the rubber band (lungs) and the hanger.

We have found that using this model during discussion of the separate relaxation curves for the lung and chest cage, as well as the combined lung-chest relaxation curve (Fig. 4), greatly enhances the student’s understanding (parenthetically, how many of our students really reach an understanding of these curves?). Students are encouraged to manipulate the model as the instructor describes the specific components shown in Fig. 4.

Alveolar Volume Model

Once the dynamics of the lung and chest wall are understood, we ask our students, “What is alveolar ventilation?” As you know, alveolar ventilation is the volume of fresh air introduced into the gas-exchanging regions of the lungs per minute. This concept, seemingly straightforward, is confounded and confused by two important features: 1) anatomic dead space and 2) “fresh” air.

To help unconfound and unconfuse this concept, students are told that at the end of a normal expiration (just before the next inspiration) the conducting airways (anatomic dead space) are filled with alveolar gas. Thus, as a tidal inspiration begins, the alveoli must first receive the gas that was in the anatomic dead space from the last exhalation. This gas does not raise alveolar Po2 or lower alveolar PCO2 very much because it has the same composition as the alveolar gas. After the dead space volume is inspired, the alveoli receive fresh air until the tidal volume is completed. The last portion of the fresh air, of course, remains in the conducting airways.

The model used to demonstrate this is shown in Fig. 5, which shows a paper cutout of the anatomic dead space with the alveoli as well as two columns of paper with four segments, with each segment representing 150 ml of air (top). As shown in Fig. 5, top, one column has three 150-ml segments (dark blue boxes) and one 150-ml segment (light blue box), and the second column has three 150-ml segments (light blue boxes) and one 150-ml segment (dark blue box). The dark blue segments represent atmospheric (fresh air) and light blue segment represent alveolar air (air that has previously equilibrated with pulmonary artery blood) (Fig. 5, top). The anatomic dead space and columns of air are sized such that one of the 150-ml segments fits perfectly into the anatomic dead space while the remaining three segments fit perfectly into the alveoli (Fig. 5, bottom).

To use the model, we start at the end of a normal expiration (just before the next inspiration) when the conducting airways are filled with alveolar gas. At this point, the students place the column of air with one light blue segment into the anatomic dead space and the three dark blue segments projected into the atmosphere. Thus, as a tidal inspiration begins (and the students slide the column of air into the alveoli), the alveoli must first receive the gas that was in the anatomic dead space from the last exhalation (alveolar air, light blue segment). After the dead space volume is inspired, the alveoli receive fresh air (atmospheric air, dark blue segments) until the tidal volume is completed. The last portion of the fresh air, of course, remains in the anatomic dead space (dark blue segment). Thus, three segments of fresh air (450 ml) entered the anatomic dead space, but only two segments of fresh air entered the alveoli. Since alveolar volume is only the volume of fresh air entering the alveoli, alveolar volume is 300 ml. That is, during inspiration, tidal volume is 450 ml. Since the dead space is 150 ml, this means that the alveolar volume (the volume of fresh air reaching the airspaces) is 300 ml.

At the end of a normal inspiration (just before the next expiration), the conducting airways are filled with fresh atmospheric air. At this point, the students place the column of air with one dark blue segment (fresh atmospheric air) into the anatomic dead space and the three light blue segments into the alveolus (old alveolar air; Fig. 5, bottom). Thus, as a tidal expiration begins (and the students slide the column of air into the atmosphere), the atmosphere must first receive the gas that was in the anatomic dead space from the last inspiration (fresh atmospheric air, dark blue segment). After the dead space volume is expired, the atmosphere receives alveolar air (air that had previously exchanged, light blue segments) until the tidal volume is completed. The last portion of the fresh air, of course, remains in the anatomic dead space (light blue segment). Thus, three segments of alveolar air (450 ml) entered the anatomic dead space, but only two segments of alveolar air entered the atmosphere. Although this concept is confounded by the anatomic dead space and confused (not necessarily complex) because of the “fresh” air concept, the model significantly helps our students grasp the concepts.

Distribution of Alveolar Ventilation Model

Up to this point, we have air entering and exiting the lungs; however, where does it go? To answer this question, we discuss the distribution of alveolar ventilation and the effect of gravity. The effect of gravity on the distribution of ventilation within the lung is very important since this has implications for the matching of ventilation to perfusion in both normal and pathological lungs as well as during one-lung ventilation for thoracotomy procedures.

Therefore, we emphasize that, because of gravity, there are unique compliance characteristics for different alveoli ranging from the base to the apex of the upright lung. The alveoli at the top of the lung are ventilated less than those at the bottom. The reason is that the alveoli at the top are on a less compliant part of the compliance curve, e.g., they are already “stretched,” and,
for any given pressure change, they will expand less than their counterparts at the base.

At this point, it is important to remind students that the directionally opposite recoil forces of the lung and chest wall (the tug of war) create an intrapleural pressure that is below atmospheric pressure [the tug of war between the lung and chest wall increases the intrapleural space and decreases the intrapleural pressure (13)]. Furthermore, as a person inhales to expand the chest cage and lung, intrapleural pressure becomes increasingly subatmospheric as a result of the force tending to separate the lung and chest wall. Thus, because the alveoli at the top of the lung are stretched (greater force tending to separate the lung and chest wall), the intrapleural pressure is more negative at apical alveoli and transmural pressure (intra–alveolar minus intrapleural pressure) is greater due to an expanded apical alveoli.

To model this concept, the students are given a small Slinky (Fig. 6) and are told that the lungs are like a Slinky, hanging from the ceiling (11). The coils closest to the ceiling are wide apart, already stretched, and therefore on the end of the compliance curve. In contrast, the coils at the bottom are very close together, compressed, and therefore on the steep part of the compliance curve. The students are encouraged to consider how this effects regional distribution of air flow during inspiration.

Specifically, because of the way in which the lungs are suspended in the chest cavity and are subjected to gravity, a gradient in pleural pressure exists from the apex to the base of ~7.5 cmH₂O. Intrapleural pressure surrounding apical alveoli is more negative than intrapleural pressure surrounding basal alveoli. Thus, transpulmonary pressure is greater for apical alveoli because these alveoli are “stretched” open. If a subject starts inspiration from FRC, apical alveoli will start off at a larger volume than basal alveoli due to the more negative intrapleural pressure; thus, apical alveoli will be less able to accommodate any increase in volume (i.e., they are less compliant than basal alveoli). Thus, if inspiration starts from FRC, most of the incoming air goes preferentially to basally located alveoli, which started off less distended and thus more compliant.

The Slinky perfectly illustrates this concept because the coils at the top are already stretched, and, for any given pressure change, these coils will expand less than their counterparts at the base, which are not stretched. In addition, using this model, students are able to see the changes in FRC associated with the supine position.

Discussion

When teaching and learning about alveolar ventilation with our class of 300 first-year medical students, we use four simple, inexpensive models. We (my graduate students and I) construct the models and place the models into plastic bags. I believe that this is an important lesson for graduate students. Our graduate students must understand that teaching is no less difficult than research (25) and classes must be planned as carefully and thoroughly as experiments. This includes reading the educational research, designing and testing equipment (materials), and planning activities. The models are distributed to the students before class, and these activities are conducted in a very large lecture room. The students expect something new every day, which generates energy, excitement, and motivation. The models encourage thinking about the complex interactions, and students appreciate manipulating the objects and actually understanding how they work. Using models also allows us to show students how we think as well as what we know. Finally, students enjoy taking the models home to demonstrate to friends and family “how the body works” as well as use the models as future study aids.

We believe that it is important to plan an activity for students because activities engage the audience, build curiosity and create a challenge. That is, learning is not a spectator sport, and students are not vessels passive ly waiting to be filled with a predetermined body of knowledge. Students do not learn by simply sitting in a classroom listening to the teacher, memorizing prepackaged assignments, and spitting out answers (3). Students learn when they are actively involved in learning (16). Students must do more than just listen: they must read, write, discuss, and be engaged in solving problems (3). Remember, most of the learning occurs outside the classroom when students read, reflect, write, or experience the information. Models support these activities as well as provide elements of surprise and concrete examples.

REFERENCES